The Contribution of Locally Grown Foods in Cumulative Exposure Assessments

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ABSTRACT:

Both laboratory and field studies confirm the importance of vegetation for scavenging semi-volatile organic chemicals (SVOCs) from the atmosphere and a number of exposure studies have found that the dietary pathway is often a significant contributor to cumulative exposure for these chemicals. Exposure calculations based on published concentration data for foods indicate that the potential intake through ingestion is up to 1000 times that of inhalation for several persistent SVOCs. However, little information exists on the source-to-dietary intake linkage for SVOC's. Because of higher SVOC emissions to urban regions, this linkage is particularly important for foods that are grown, distributed and consumed in or near urban regions. The food pathway can also contribute to dietary exposure for populations that are remote from a pollutant source if the pollutants can migrate to agricultural regions and subsequently to the agricultural commodities distributed to that population. We use the characteristic travel distance (CTD) and available data within the CalTOX multimedia model framework to assess the contribution of local food markets to the fraction of cumulative food intake that is attributable to local sources. For a set of three representative multimedia SVOCs-benzo(a)pyrene, fluoranthene, and 2,3,7,8-TCDD, we explore the contribution of airborne SVOC's to cumulative uptake through the local food consumption pathway. We use the population based intake fraction (iF) to determine how SVOC intake varies among food commodities and compares to inhalation. The approach presented here provides a useful framework and starting point for source-to-intake assessments for the air-to-dietary exposure pathway.

INTRODUCTION

There is a growing demand for methods to assess exposure to toxic chemicals via ingestion of food, motivated in large part by the U.S. Food Quality Protection Act of 1996 (FQPA) (EPA, 1996). The FQPA requires that EPA consider aggregate and cumulative exposure to pesticide residues in food. This has prompted exposure scientists to develop methods for combining measured residues in food products or raw agricultural commodities with food consumption rates to estimate dietary exposure, using tools such as the Dietary Exposure Evaluation Model, DEEM (Barraj et al., 2000), or the Dietary Exposure Potential Model, DEPM (Tomerlin et al., 1997; EPA, 2000).

However, combining residue data with food consumption data provides little insight about source-to-intake relationships for dietary exposures to other environmental pollutants, particularly the semivolatile organic chemicals (SVOCs) that partition from environmental media into the agricultural foodchain. Earlier literature on food pathway models such as the PATHWAY model for radionuclides (Whicker and Kirchner, 1987), provide an explicit treatment of the full pathway from air to final exposure concentrations in food products. For SVOCs there is a need for a similar evaluative framework that includes explicit quantification of the links between air emissions and food consumption.

A number of SVOC's, including polycyclic aromatic hydrocarbons (PAH's), and polychlorinated dibenzo-dioxins and furans (PCDD/F's), are important contributors to potential ecological and/or human health risk through chronic exposures (Guillen et al., 1997; EPA, 2001a; EPA, 2001c). These chemicals are released primarily as combustion by-products into the atmosphere from point- and area- sources, on- and off-road mobile sources, and via resuspension and remobilization of dust particles with sorbed SVOCs

due to developments associated with urban sprawl (VanMetre et al., 2000). In addition, natural sources such as forest fires and volcanic eruptions contribute to background levels of SVOC's.

Previous work has shown that vegetation can accumulate both gas-phase and particle bound SVOCs from the atmosphere providing a means for air pollutants to enter the foodchain (Hattemer-Frey and Travis, 1991; Jones et al., 1991; EPA, 1994; McLachlan, 1996; Welsch-Pausch and McLachlan, 1998; EPA, 1998d; Bohme et al., 1999; Kaupp et al., 2000). Plants also accumulate pollutants from the soil but the importance of the soil-to-plant pathway for atmospheric pollutants is generally negligible (Welsch-Pausch et al., 1995). Plants grown in or near an urban region have significantly higher PAH concentrations than rural plants (Wagrowski and Hites, 1997) indicating that the proximity of vegetation relative to the source of pollutant is important. McKone reports that human exposures due to vegetation are one of the most uncertain exposure pathways in multimedia fate and exposure models (McKone, 1994). This is in part due to the complex kinetics involved in transfers from air to plants, to variation in cuticle chemistry and plant architecture (i.e., horizontal surface area / plant volume ratio), and to difficulties in relating the level of contamination in raw agricultural commodities to exposure concentrations in food products.

Although there is a large degree of uncertainty in relating environmental concentrations to dietary intake, exposure surveys indicate that the diet is a dominant exposure pathway for a number of persistent SVOCs. For example, using food consumption databases along with measured and estimated levels of dioxins in food products, the US EPA (EPA, 2001a) estimates that agricultural products, i.e., dairy,

meat, produce, eggs, and fish, contribute approximately 97% to dioxin intake in the US population. Only 2.5% of intake is attributable to inhalation, and even less to drinking water consumption, non-dietary ingestion, and dermal contact with contaminated soil (EPA, 2001a). Measured concentrations in foods also reveal that dietary intake can be significantly greater than inhalation for PAHs (Lioy et al., 1988; Butler et al., 1993; Chuang et al, 1999; Wilson et al., 2001).

The absence of reliable models for dietary source-to-intake pathways means that food exposure assessments must be based primarily on chemical residue data. We list representative chemical residue databases in **Table 1**. Most of the available residue databases focus on levels of pesticides, but some include data on PCBs and VOCs. Only the Fish and Wildlife Residue Database 1995 includes levels for PAH's and dioxins in cooked foods (EPA, 2000). Residue data is often obtained by collecting food from grocery stores and preparing the food according to standardized consumer recipes and then analyzing the prepared food for residues. Also, residue data has been collected from duplicate diet methods (Thomas et al., 1997). However, these methods provide only a general measure of the levels of contaminant in the bulk sample and no information about the source of chemical residue in food.

Thus, the motivation for our assessment is the failure of standard collection methodologies such as market basket studies or duplicate diet methods in combination with food intake data to supply the necessary source- to-intake information for cumulative intake models. We are further motivated by the lack of relevance of residue data for characterizing the ingestion pathway for SVOC's in cumulative, multi-pathway, multi-route exposure assessments. Because the standard data collection does not allow

for assessing the contribution of locally grown foods (including but extending beyond homegrown foods), we elected to focus on this specific issue. Approximately 34 million households have a home garden (EPA, 1997) and approximately 1 million customers in the US visit local farmer's markets each week (USDA, 1996c). The objective of this paper is to identify and evaluate the dietary source-to-intake links for SVOC's omitted to the air with particular emphasis on locally grown and consumed foods.

METHODS

Here we describe (1) how we selected chemicals for developing the exposure assessment framework, (2) the data and models used for making ingestion/inhalation pathway comparison, (3) how we identified the chemical-specific range of influence for allocating air concentrations to locally grown food products and exposed populations and, (4) the application of the intake fraction (iF) as a generalized measure of potential source-to-intake relationships.

Selection of chemicals for framework development

Among the broad class of potentially toxic SVOC's, we select three chemicals, benzo(a)pyrene, fluoranthene, and 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD), for our study of exposures through local food. Physicochemical properties of these three compounds are summarized in Table 2.

Fluoranthene is a class D compound in the EPA IRIS database (EPA, 2001b), i.e., a noncarcinogen based on no human data on carcinogenicity and inadequate animal carcinogenicity data. Fluoranthene exposures to mice led to nephropathy, increased liver weights, and hematological alterations and clinical effects (EPA, 2001b).

In the EPA IRIS database (EPA, 2001b), benzo(a)pyrene is a class B2 compound. B2 PAH's have produced positive results for DNA damage based on a variety of tests, including transformation of mammalian cells, mutations in bacteria, and positive results in genotoxicity assays. In animal studies, benzo(a)pyrene has been linked to forestomach tumors, leukemia, esophageal and laryngeal tumors resulting from oral exposures (EPA, 2001b). Thus, based on inadequate human data but adequate animal data, B2 compounds are a probable human carcinogen. In addition, the California Air Resources Board has scaled the toxicity of other B2 PAH's to benzo(a)pyrene with potency equivalence factors (PEF's) (CARB, 1997).

TCDD is a known/likely carcinogen, i.e., class A or B1 (EPA, 2001a) to which the toxicity potential of other dioxin congeners and dioxin-like compounds are related based on toxic equivalents (TEQ) methodology.

The endpoint of our assessment is potential dose as expressed by the total SVOC intake. Because of the dearth of available data on the human bioavailablilty and metabloism for these representative SVOC's, we do not quantify or compare bioavailable or metabolized doses in the present study. As a result the source-to-intake and source-to-dose relationship that we use both refer to potential dose are thus the same quantity. We therefore use these terms interchangeably..

Based on emission factors data (EPA, 1998c; McDonald et al., 2000; Cadle et al., 2001), and census data (BoC, 2000), we find that the major sources of PAH's for a typical urban region are on-road motor vehicles and wood smoke from residential heating. Using the nine county San Francisco Bay Area as a representative urban air shed, which contains and borders a multitude of sites which supply local food, we

estimate that these two sources account for approximately 10^4 kg of total PAH's /yr within this air shed. Since SVOCs emitted to the atmosphere have the potential to travel long distances (Wania and Mackay, 1996) these emissions represent a potential source of contamination to food grown in both the immediate (urban) and neighboring agricultural- air sheds.

Comparison of dietary and inhalation exposure pathways

As a first step in our approach to evaluate the contribution of the food pathway in cumulative exposure assessments, we reviewed measured levels in foods reported in the peer-reviewed literature. Since we are primarily looking at intake attributed to air-plant transfers we focus on raw, uncooked, fruits and vegetables, and grains (fvg), that are available for purchase at local food markets throughout the San Francisco Bay Area. We define the ingestion to inhalation intake ratio, $\theta_{ing/inh}$(fvg), for a compound as

$$\theta_{ing/inh}(fvg) = \frac{\sum \overline{C_{j,w}} \times IR_j}{C_{air} \times BR} \qquad \text{(Equation 1)}$$

where, $\overline{C_{j,w}}$ is the intake-weighted concentration of a chemical in food category j, μg/kg (see appendix, Part A, for how $\overline{C_{j,w}}$ was calculated); IR_j is the intake of particular food category, j, kg(j)/kg(BW)/d; C_{air} is the concentration of the chemical in air, μg/m^3; BR is the breathing rate, m^3/kg(BW)/d.

Because the magnitude of the ingestion/inhalation intake ratio is well established for TCDD and because of limited available data for concentrations in raw fvg's for fluoranthene, we focus our comparison on benzo(a)pyrene. We consider the following seven food categories (i.e., j= 7): (1) exposed and (2) protected fruits; (3) exposed and

(4) protected vegetables; (5) root and (6) leafy vegetables; and (7) grains. We use the term "exposed" to refer to commodities that have direct contact with atmospheric deposition, whereas for "protected" commodities, the consumed portion is has no direct air contact. For C_{air} values, we used activity-weighted indoor and outdoor concentrations from measured concentrations published in the peer reviewed literature and reports and time-activity budgets from the National Human Activity Pattern Survey (Klepeis et al., 2001), to obtain a distribution of the time-weighted average concentration for an individual in the exposed population. Table 3 provides summary statistics of each of the parameters in Equation 1.

Measurements of SVOCs in foods are limited, but, benzo(a)pyrene concentrations are reported for the widest range of fvg types relative to other PAH's. Table 3(a) reflects a possible range of benzo(a)pyrene levels in fvgs and the summary statistics may be considered as a lower bound on the potential range since uncertainties, such as measurement error, are not included in these ranges. For each of the parameters in Equation 1, we used the arithmetic mean and standard deviation values to construct lognormal distributions of the relevant parameters for estimating $\theta_{ing/inh}(fvg)$. Because the data for these parameter values came from different sources, we did not assume a correlation between concentrations in air and fvg's. We performed a Monte Carlo analysis (n=1000 trials) with Crystal Ball 2000 (Decisioneering, 2000) to generate a distribution of $\theta_{ing/inh}(fvg)$ values. These $\theta_{ing/inh}(fvg)$ ranges are compared with estimates of $\theta_{ing/inh}(fvg)$ obtained from the CalTOX multimedia exposure model (McKone et al., 2002). The CalTOX model calculates ingestion intake on a fresh weight basis, does not account for indoor sources, and includes grains as unexposed produce.

We adjusted the CalTOX model so that 100% of the fruits, vegetables, and grains consumed came from a region with the same C_{air} used for the inhalation concentration. In addition, for the CalTOX comparisons, the local food contribution was set at 100% ("unit world") and Bay area (1.87E10 m^2) by California landscape parameters and residential exposure factors.

CalTOX multimedia exposure model

CalTOX was developed as a set of spreadsheet models and spreadsheet data sets to assist in assessing human exposures from multiple environmental media through multiple pathways. CalTOX was originally developed for the California Environmental Protection Agency's Department of Toxic Substances Control to assist in the process of setting soil clean-up standards at uncontrolled hazardous waste sites (McKone, 1993). We used CalTOX version 4.0 (beta) (McKone et al., 2002) which is an eight-compartment regional and quasi-dynamic multimedia mass-balance model. The eight CalTOX compartments are (1) air, (2) plant surfaces (cuticle) (3) plant leaf biomass (leaves), (4) ground-surface soil, (5) root-zone soil, (6) the vadose-zone soil below the root zone, (7) surface water, and (8) sediments. CalTOX includes models of 23 different exposure pathways that link the environmental media concentrations to ingestion, inhalation, or dermal intake.

In the CalTOX framework, environmental concentrations are derived by determining the likelihood of competing processes by which chemicals (a) accumulate within the compartment where they are released, (b) are physically, chemically, or biologically transformed within the source compartment (i.e., by hydrolysis, oxidation, etc.), or (c) are transported to other compartments by cross-media transfers that involve

dispersion or advection (i.e., volatilization, precipitation, etc.). Exposure modeling is

based on transfer factors that determine the concentration in exposure media such as

indoor air, fruits, vegetables, grains, meat, dairy products, eggs, and drinking water. For

example, CalTOX calculates the potential long term daily intake via ingestion of an

organic chemical as the sum of the *contaminant contact rate for a given food [kg food /*

kg-BW / d] x Concentration in that food [μg / kg food]. The concentration in each food

type is estimated from the mass balance or using measured or estimated

bioconcentration factors along with estimated environmental concentrations. Pathways

analyzed in the CalTOX mutlimedia modeling framework are shown in Figure 1. The

algorithms used to estimate route-specific potential dose in the CalTOX model are

described in detail elsewhere (McKone, 1993).

**Allocating air concentrations to food products and exposed populations-Issues
of Scale**

To determine SVOC ingestion intake resulting from air emissions we consider

two levels of scale, the environmental reach of an SVOC, and the scale of food

distribution. We define the scale of food distribution as the distance that food travels

from the point of production (and potential contamination) to the point of purchase/

consumption. In this study, we focus on the farm-to-market travel distance for local

foods. We define the reach of an SVOC as the characteristic travel distance (CTD) of

the pollutant, which is a screening level measure of the distance that a pollutant travels

in the environment from a release region (Bennett et al., 1998).

We use the San Francisco Bay Area as a case study for the local food travel

distance. We estimated farm-to-local market distances from a database of addresses

(Silveira, 1999) for three San Francisco Bay Area farmer's markets operating in the Spring of 1999, i.e., Berkeley (n = 50 farmers/vendors), Pleasanton (n= 44 farmers/vendors), and Oakland (n =48 farmers). Farm-to-market radial distances were estimated (Silveira, 1999) using Streets98 software (Microsoft, 1997). For overlapping vendors that sold their products at more than one Farmer's market, we used the average of their distances (n= 109 distinct farmers).

The farm-to-market distance was compared with the CTD of the pollutant to assess the local food contamination potential for a given SVOC. The CTD is the radial distance from the source where the concentration falls to 37% of the original value (Bennett et al., 1998). The CTD, in simplified form is calculated as:

$$CTD= u / k_{eff} \qquad \text{(Equation 3)}$$

Where u is the mean long-term average wind speed, m/s, and k_{eff} is the effective reaction rate of a chemical, s^{-1}, in the environment based on multimedia dispersion and transformation processes. As proposed and applied by Bennett and others (Bennett et al., 1998; Bennett et al., 2000; Bennett et al., 2000) k_{eff} takes into consideration partitioning and degradation of a chemical in air, soil (three layers including surface, root zone, and vadose), and water (surface and ground). We used the CalTOX multimedia modeling framework, as described above parameterized with California landscape parameters, to calculate a distribution of CTD's.

The Intake Fraction (iF)

We use the concept of intake fraction (iF) to represent the strength of the source-to-intake relationship for different pathways, k, linked to air emissions, including food (e.g., air-to-fruit, air-to-feed-to-meat/milk, etc), as well as inhalation and dermal

pathways. The iF has been introduced as a convenient population-scale, surrogate

measure for assessing potential exposure, or the "effectiveness of delivery from the

source to the target" (Zartarian et al., 1997). We use the population-based iF,

formulated here as (Bennett et al., 2002):

$$iF(k) = \frac{Intake_k[kg/d]}{emission[kg/d]} \rightarrow \frac{Intake_k(individual)[kg/d-person]}{emission[kg/d]} \times population \qquad \text{(Equation 4)}$$

where the $Intake_k$ is the total intake quantity through pathway k by all exposed

individuals (kg d^{-1}); emissions are the total amount released to air, also in kg d^{-1}

$Intake_k(individual)$ is the potential long-term average daily individual intake by pathway k

(kg d^{-1}), as obtained from either measurements or a model such as CalTOX; and,

population is the number of individuals represented by the $Intake_k(individual)$. The

challenge of the iF assessment is to determine how to link an emission to the

appropriate population. Thus, pollutant transport scale and food distribution scale are

key inputs to this assessment.

We used the CalTOX multimedia model framework, as described above, to

calculate a range of iF values for the pathways shown by Figure 1, and link these to

intake by inhalation, ingestion, and dermal routes. As noted above our range of iF

values, were obtained by applying CalTOX to the San Francisco Bay Area, which we

modeled as a population of 7 million people uniformly distributed across a 18700 km^2

(~105km x 190km) total land (+ surface water) region, with an atmospheric mixing

height of 700 m, and average meteorological and landscape parameters for the state of

California (McKone et al., 1998b). Relevant parameters needed to calculate the intake

fraction for all of the pathways shown in Figure 1 were assigned lognormal distributions,

as described in (McKone, 1993). We used the same modeling scenarios and

assumptions as for the $\theta_{ing/inh}$(fvg) described above, under Methods. Thus, the fraction

of local foods was set at 100% as a bounding analysis, so we could assess the potential

contribution of local foods to total intake of SVOC's. SVOC source emissions were set

at 1 mol/d to air.

Results and Discussion

Comparison of dietary and inhalation exposure pathways

Using measurements of food intake and PAH food concentrations from the

literature, we developed a distribution of $\theta_{ing/inh}$ (fvg)s for benzo(a)pyrene as shown in

Figure 2. The range of ingestion-to-inhalation benzo(a)pyrene intake ratios generally

falls between 0.1 and100 but can be as high as 1,000. It should be noted that in

comparison with other published breathing rate values, our values are at the high end,

and may even be a factor of two higher (Layton, 1993). If we used smaller BR's this

would only increase $\theta_{ing/inh}$.

As illustrated in Figure 2, below the 75th percentile of the results, we found good

correspondence between the $\theta_{ing\,/\,inh}$(fvg) from the CalTOX model and the $\theta_{ing\,/\,inh}$(fvg)

derived from the literature values summarized in Table 3 when we assume that the

contribution of local foods comes from a distance of 3 CTD's . When we consider that

all of the fvgs come from within the source region, i.e., the San Francisco Bay Area, we

capture the upper end of the empirical data distribution, as shown in Figure 2. Thus, the

empirical data is bounded at the lower end by the model assuming fvgs originate within

3 CTDs and at the upper end assuming all fvgs are grown within the source region. This

also suggests that we cannot use a single model scenario as represented by either of

the two CalTOX versions displayed in Figure 2, to capture the range of source-to-intake pathways that exist in the US.

Our method for calculating $\theta_{ing/inh}$, can also be applied to other SVOCs to evaluate the range of ingestion intake relative to inhalation. However, data for determining concentrations in air, raw fruits, vegetables and grains for other SVOC's is sparse. Further, extending $\theta_{ing/inh}$(fvg) to include meats and dairy products (such as eggs and milk) is not currently possible since measured concentrations attributable to ambient environmental levels are extremely limited for these food products.

Allocating air concentrations to food products and exposed populations: Issues of Scale

In assessing local food exposures to airborne SVOC's, there are two central issues related to scale that must be resolved before we can begin to characterize source-to-intake relationships beyond inhalation in cumulative exposure assessments. First, we must determine how far "local" food travels from the point of production to consumption. That is, does the term "local" (including homegrown) imply that food is grown in the same airshed as the source? Second, we must determine how far from the point of release a chemical travels in the environment.

The leftmost boxplot in Figure 3, shows that farm-to-local market distances for food products transported to farmers' markets in the San Francisco Bay Area is generally between 80-150 km (the inter-quartile range, IQR), with a median of 106km. In the multimedia and exposure modeling literature, local has been reported as a 10-20 km distance from pollutant/contaminant source (Mackay and Webster, 1998) and the EPA has typically assumed a 50 km radius from the source for air dispersion modeling (EPA,

1998a; EPA, 1998b). However, little scientific justification has yet been provided for these ranges.

In comparison, the farm to local market distance that produce travels to the San Francisco Bay Area is nearly double the national average found by the USDA (USDA, 1996c). Of the farmers surveyed by the USDA, 95% travel less than 56 km to bring their crops to local farmer's markets (USDA, 1996c). The Leopold Center (Leopold Center, 2001) reports the weighted average source distance for local produce (en route to local farmers markets) is approximately 100 km. For the San Francisco Bay Area, much of the food) arrives from the Central and Salinas Valley agricultural regions (~ 3/5ths of all farmland in California). Figure 4 displays where the San Francisco Bay Area is situated with respect to the Central and Salinas Valley agricultural regions. As can be seen from Figure 4, the San Francisco Bay Area is within the farm-to-local market travel distance, shown by the leftmost boxplot of Figure 3, of most of the farmland in California.

Ranges of CTD's for the three representative SVOCs are also given to the right of the farm-to-market distance in Figure 2. We can make several conclusions based on the range of CTD's and farm-to-market distances, all of which uphold the importance of including the contribution of local foods in cumulative exposure assessments. For the San Francisco Bay Area, the range of farm-to-local market distances are on the same order of magnitude as the CTD for the two PAH's. However, for dioxin, the levels found in local food cannot be differentiated between local and regional pollution sources. Indeed, the median range of the CTD for dioxin is on the same order of magnitude as the conventional (i.e., farm-to-grocery store) travel distance of food of approximately

2000 km (Leopold Center, 2001). This implies that food exposures to dioxin can be adequately assessed by simply combining residue and intake data.

Our results indicate that local foods can be contaminated by SVOCs released to air, depending on the chemical specific properties of the SVOC. When used to plan a cumulative exposure assessment, a pollutant's CTD can help identify which SVOC's are more likely to contribute to adverse health risk via chronic consumption of locally produced foods. Also, the CTD can help characterize the potential size of the affected population.

A Generalized Measure of Potential Source-to-Intake Relationships

Using the iF as a source-to-intake metric allows us to relate SVOC intake through multiple exposure pathways to airborne emissions within the CalTOX multimedia modeling framework. For the two PAH's that we found to have a potential impact on agricultural zones, we identified food pathways that are likely to be most important on the local scale.

The cumulative distributions of the pathway specific population iF's for benzo(a)pyrene and fluoranthene in the hypothetical scenario based on the San Francisco Bay Area Urban Region, applying the default setup of CalTOX, are presented in Figure 5 (a) and (b), respectively. We have assumed that all foods consumed are produced "locally", where local is defined by the CTD for the pollutant in the region. As shown in Figure 4, we base this assumption on the recognition of the proximity of highly productive farmlands near the San Francisco Bay Area.

Our modeled results for fluroanthene and benzo(a)pyrene, show that intake from foods is by far the largest contributor to total intake when compared with all other routes

of exposure, including dermal and inhalation. Furthermore, as the $\theta_{ing/inh}$ analysis demonstrated, fvg's dominate SVOC intake. Indeed, as Figure 5 displays, the total (all routes) iF and the ingestion (total) iF are very closely aligned. Figure 5 also shows that, as a median estimate, one out of every 10,000 molecules of benzo(a)pyrene emitted to air is consumed by this population, through fvg. For fluoranthene, we estimate that the exposed population consumes one out of every 100,000 molecules through fvg intake. The reason for this difference in iF between flouranthene and benzo(a)pyrene is most likely due to physicochemical properties. Fluoranthene is less hydrophobic than benzo(a)pyrene and thus less likely to absorb into plant surface (McLachlan, 1999).

Because of the high inter-individual variability associated with individual iF calculations and our inability to reliably capture this variability with current data and models, we use the population based iF for evaluating food pathway contributions. This provides us with a source-to-intake metric that accounts for the likely fraction of a pollutant emitted to the atmosphere that will contribute to exposure via a specified route, such as ingestion, to a human receptor. This metric reflects the potential intakes among a population of local food consuming individuals, in this case the San Francisco Bay Area. However, the ranges of the iF distributions indicate the potential range of inter-individual variability in the iF. The population based ingestion iF's vary over a much greater range than the inhalation iF's.

We cannot compare our ingestion iF's for SVOC's to the literature because published values are not available. However, the inhalation iF's calculated in this paper fall within the range published by other researchers. The inhalation iF found by Lai and coworkers (Lai et al., 2000) for a primary, relatively non-reactive, toxic air contaminant

emitted in a well mixed urban zone with a population density of 1000 people/km^2, ~ 10^{-7} to 10^{-5} of the mass emitted to ambient air would be inhaled by the population. With the San Francisco Bay Area population density of approximately 450 people/ km^2 (i.e., w/ land area ~ 15.6E3 km^2, and population 7 million) we found a range for the population based inhalation iF for benzo(a)pyrene between 10^{-8} to 10^{-6} and for fluoranthene, between 10^{-7} to 10^{-5}.

This work demonstrates the importance of including the spatial range of a pollutant and local food pathways, beyond homegrown, in cumulative exposure and risk assessments for emissions to air. Future work should focus on improving models that estimate exposure concentrations in the diet relative to environmental concentrations. This may require collection and analysis of data that includes measurements of PAH food intake and corresponding media measurements for several PAH's. There is also a need for research separating the environmental contribution of SVOC contamination in foods from the food-processing portion.

Appendix

Calculation of $\overline{C_{i,w}}$:

       We gathered individual raw fruit, vegetable and grain concentration (C_i) data from the archive literature. When a value was reported, and unless otherwise stated, it was assumed that the concentration referred to the mean value of the measured samples. Where individual fruit and vegetable raw C values were available (rarely), we calculated an arithmatic mean and standard deviation of the reported values. All individual non-detects (NDs) from the literature were assumed to be zero in the $\theta_{ing/inh(fvg)}$ analysis.

       Of 152 reported C_i values, 41 were reported on a fresh weight (FW) basis, 63 were reported on a dry weight (DW) basis, and 48 were reported without designation as to FW or DW. All DW C_i values, were converted to FW by the following conversion:

       $C_{FW} = C_{DW} \times [\,(100 - W) / 100\,]$

Where, W is the mean moisture content (% of edible portion) of individual raw fruits, vegetables and grains (EPA, 1997), Table 9-27 for fruits and vegetables; Table 12-21 for grains).

       For the undesignated C_i values, we calculated a midrange value, assuming the reported concentration was reported on a FW and DW basis.

       If more than one value was reported or calculated for a specific raw fruit, vegetable or grain, C_i multiple values were averaged to give $\overline{C_i}$. By taking, $\overline{C_i}$, we realize that averaging averages might under-specify the true standard deviation (range) of the distribution of raw fruit, vegetable and grain concentrations. The final C_i , or $\overline{C_i}$ values are summarized in Table A-1.

For each of the j's (i.e., six f&v categories, and grains) the C_i, or if available, the $\overline{C_i}$, values were weighted with respect to the intake of each i, for an intake averaged concentration, $\overline{C_{j,w}}$, calculated as:

$$\overline{C_{j,w}} = \frac{\sum_{i=1}^{n} w_i \times \overline{C_i}}{\sum_{i=1}^{n} w_i}$$

where, n is the total number of i's (i.e., individual fvg types) within each j; w_i are the intake weights, on an 'as-consumed mean per capita' basis (EPA, 1997)Table 9-13 and Table 12-12). We assume here that 'as-consumed' is equivalent to FW; $\overline{C_i}$ are the individual fvg concentrations, as summarized in Table A-1.

We also calculated a weighted standard deviation (sd_w) for the $\overline{C_{j,w}}$'s, as:

$$sd_w = \sqrt{\frac{\sum_{i=1}^{n} w_i (\overline{C_i} - \overline{C_{j,w}})^2}{\frac{(N'-1)\sum_{i=1}^{n} w_i}{N'}}}$$

where, n is the amount of individual fruit and vegetable or grain types (i) for each category, j; N' is the amount of nonzero weights (i.e., the number of non-zero intakes, w_i); $\overline{C_i}$ is the non-intake weighted concentrations of the i's; $\overline{C_{j,w}}$ are the intake-weighted mean concentration of the specific fvg category, j; w_i is the intake weight for the ith fruit, vegetable, or grain observation, on an 'as-consumed mean per capita' basis (EPA, 1997); Table 9-13 and Table 12-12). We assume here that 'as-consumed' is equivalent to FW.

References

Baek, S, R Field, M Goldstone, P Kirk, J Lester and R Perry. A Review of Atmospheric Polycyclic Aromatic Hydrocarbons: Sources, Fate, and Behavior. *Journal of Water, Air, and Soil Pollution* 1991; 60: 279-300.

Barraj, L, B Petersen, J Tomerlin and A Daniel. Background Document for the Sessions: Dietary Exposure Evaluation Model (DEEM) and DEEM Decompositing Procedure and Software. *Presented by: Novigen Sciences, Inc. Washington, DC and the United States Environmental Protection Agency (US EPA), Office of Pesticide Programs, Washington, DC. Presented to: FIFRA Scientific Advisory Panel (SAP), Arlington, Virginia February 29 – March 3, 2000* 2000; Available at: http://www.epa.gov/scipoly/sap/2000/february/Final_sap_document_Feb_1_2000.pdf. Last accessed January 2002.

Bennett, D, T McKone, J Evans, W Nazaroff, M Margni, O Jolliet and K Smith. Defining Intake Fraction. *Environmental Science and Technology* 2002; 3A-7A.

Bennett, D, T McKone and W Kastenberg. Characteristic Time, Characteristic Travel Distance, and Population Based Potential Dose in a Multimedia Environment: A Case Study. *LBNL Report-45815, Environmental Energy Technology Division.* 2000a.

Bennett, D, T McKone and W Kastenberg. Characteristic Time, Characteristic Travel Distance, and Population Based Potential Dose in a Multimedia Environment: A Case Study. *To be published in: Human and Ecological Risk Assessment: Theory and Practice, John Wiley and Sons, New York, 2000b.*

Bennett, D, T McKone, M Matthies and W Kastenberg. General Formulation of Characteristic Travel Distance for Semivolatile Organic Chemicals in a Multimedia Environment. *Environmental Science and Technology* 1998; 32(24): 4023-30.

BoC. Bureau of Census, Census 2000 data. A*vailable at http://www.census.gov. Last accessed November, 2001.* 2000; .

Bohme, F, K Welsch-Pausch and M McLachlan. Uptake of Airborne Semivolatile Organic Compounds in Agricultural Plants: Field Measurements of Interspecies Variability. *Environmental Science and Technology* 1999; 33(11): 1805-1813.

Butler, J, G Post, P Lioy, M Waldman and A Greenberg. Assessment of Carcinogenic Risk from PErsonal Exposure to Benzo(A)pyrene in the Total Human Environmental Exposure Study (THEES). *Journal of Air and Waste Management Association* 1993; 43: 970-77.

Cadle, S, P Mulawa, P Groblicki and C Laroo. In-Use Light-Duty Gasoline Vehicle Particulate Matter Emissions on Three Driving Cycles. *Environmental Science and Technology* 2001; 35(1): 26-32.

CARB. Toxic Air Contaminant Identification List Summaries- ARB/SSD/SES. *Section 9: Particulate Organic Matter* 1997; 1997.

Chuang, JC, PJ Callahan, CW Lyu, NK Wilson. Polycyclic Aromatic Hydrocarbon Exposures of Children in Low-income Families. *Journal of Exposure Analysis and Environmental Epidemiology.* 2:85-98. 1999.

Coleman, P, R Lee, R Alcock and K Jones. Observations on PAH, PCB, and PCDD/F Trends in UK Urban Air, 1991-95. *Environmental Science and Technology* 1997; 31(7): 2120-24.

Decisioneering. Crystal Ball Standard Edition 2000.2. 2000.

DPR. California's Residue Monitoring Program, 1986-2000. *Cal EPA, Department of Pesticide Regulation (DPR). Data available for download at: http://www.cdpr.ca.gov/docs/pstrsmon/rsmonmnu.htm. Last accessed January 2002* 2000.

Edwards, N. Polycyclic Aromatic Hydrocarbons (PAH's) in the Terrestrial Environment- A Review. *Journal of Environmental Quality* 1983; 12(4): 427-441.

EPA. Enhancement of the Pesticide Residues Information System. *US Environmental Protection Agency's Office of Policy, Planning and Evaluation (OPPE). Vol I and II, NTIS number PB93-209013. & PB93-209021.* 1993.

EPA. Estimating Exposure to Dioxin-Like Compounds. Volume II: Properties, Sources, Occurrence, and Background Exposures. External Review Draft . 1994; Office of Research and Development. Washington, DC 20460, EPA/600/6-88/005Cb.

EPA. Food Quality Protection Act of 1996. Public Law 104-170, August 3, 1996. *Located at: http://www.epa.gov/oppfead1/fqpa/gpogate.pdf. Last accessed January 2002.* 1996.

EPA. Exposure Factors Handbook. Office of Research and Development. National Center for Environmental Assessment. Washington DC. PB98-124217. Located at http://www.epa.gov/ncea/exposfac.htm. Last accessed November, 2001. 1997.

EPA. Urban Air Toxics Report. Appendix B: Modeled Outdoor Concentrations of HAP's: Analysis of Data from the Cumulative Exposure Project for the Urban Area Source Program. Office of Policy, Planning and Evaluation, Office of Air Quality Planning and Standards. May 1998. 1998a.

EPA. Study of HAP Emissions from Electric Utility Steam Generating Units- Final Report to Congress Vol I, EPA Report No. 453/R-98-004. Office of Air Quality Planning and Standards. 1998b.

EPA. Locating and Estimating Air Emissions from Sources of Polycyclic Organic Matter. *US EPA Office of Air Quality Planning And Standards. EPA-454/R-98-014. July 1998. Available at: http://www.epa.gov/ttn/chief* 1998c.

EPA. Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions- Update to EPA/600/6-90/003 Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions. *EPA 600/R-98/137.* 1998d.

EPA. Dietary Exposure Potential Model Version 3.3.2, April 200. *Program developed for US EPA National Exposure Research Laboratory, Cincinnati OH by Novigen Sciences, Inc., Washington, DC and Environ Life Sciences, Arlington, VA. Program available at: http://www.epa.gov/nerlcwww/depm.htm* 2000; last accessed January, 2002.

EPA. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds (DRAFT). *Rpt No. EPA/600/P-001*. Available at: http://www.epa.gov/ncea/dioxin.htm. Last Accessed January 2002. 2001a.

EPA. Integrated Risk Information System (IRIS). US EPA, National Center for Environmental Assessment. Located at http://www.epa.gov/iris/index.html. Last accessed November 2001. 2001b.

EPA. Urban Air Toxics Program, List of 34. *Located at: http://www.epa.gov/ttn/uatw/urban/list33.html.* Last accessed September, 2001. 2001c.

FDA. FDA Pesticide Program Residue Monitoring 1993-1999. *Pesticide Program: Residue Monitoring Reports* 2000; US Food and Drug Administration, Center for Food Safety and Applied Nutrition, April 2000. Data available for download at http://vm.cfsan.fda.gov/~dms/pesrpts.html. Last accessed June 2002.

FDA. Total Diet Study. US Food and Drug Administration Center for Food Safety and Applied Nutrition, Office of Plant and Dairy Foods and Beverages, April 2001. Data a*vailable for download at: http://vm.cfsan.fda.gov/~comm/tds-toc.html.* Last accessed January, 2002.

FSIS. Microbiological and Residue Computer Information Systems (MARCIS) by the Food Safety and Inspection Service/USDA. Published summaries available to public. Raw data available to other government agencies through FSIS. Joanne Hicks, FSIS, (Phone: 202-501-6354). 1995.

Grimmer, G and A Hildebrandt. Kohlenwasserstoffe in der Umgebung des Menschen. II. Der Gehalt polycyclischer Kohenwasserstoffe in Brotgetreide verschiedener Standorte. *Zeitschrift fur Krebsforschung* 1965a; 67: 272-77.

Grimmer, G and A Hildebrandt. Kohlenwasserstoffe in der Umgebung des Menschen. III. Der Gehalt polycyclischer Kohlenwasserstoffe in verschiedenen Gemusesorten und Salaren. *Deutsche Lebensmittel-Rundshcau* 1965b; 61: 237-39.

Guillen, M, P Sopelana and M Partearroyo. Food as a Source of Polycyclic Aromatic Carcinogens. *Reviews of Environmental Health* 1997; 12(3): 133-46.

Hattemer-Frey, H and C Travis. Benzo(a)pyrene: Environmental Partitioning and Human Exposure. *Toxicology and Industrial Health* 1991; 7(3): 141-157.

Hawthorne, S, D Miller, J Langenfeld and M Krieger. PM-10 High Volume Collection and Quantitation of Semi- and Nonvolatile Phenols, Methoxylated Phenols, Alkanes, and Polycyclic Aromatic Hydrocarbons from Winter Air and Their Relationship to Wood Smoke Emissions. *Environmental Science and Technology* 1992; 26(11): 2251-62.

IARC. IARC Monographs on the Evaluation of Carcinogenic Risk of the Chemical to Man: Certain polycyclic aromatic hydrocarbons and heterocyclic compounds. V 3. International Agency for Research on Cancer, Lyon, France. 1973.

Jones, K, T Keating, P Diage and A Chang. Transport and Food Chain Modeling and its Role in Assessing Human Exposure to Organic Chemicals. *Journal of Environmental Quality* 1991; 20: 317-29.

Kaupp, H, M Blumenstock and M McLachlan. Retention and mobility of atmospheric particle-associated organic pollutant PCDD/Fs and PAHs in maize leaves. *New Phytologist* 2000; 148: 473-80.

Kazerouni, N, R Sinha, C Hsu, A Greenberg and N Rothman. Analysis of 200 food items for benzo(a)pyrene and estimation of its intake in an epidemiologic study. *Food and Chemical Toxicology* 2001; 39: 423-36.

Kipopoulou, A, E Manolli and C Samara. Bioconcentration of polycyclic aromatic hydrocarbons in vegetables grown in industrial area. *Environmental Pollution* 1999; 106: 369-80.

Klepeis, N, W Nelson, W Ott, J Robinson, A Tsang, P Switzer, J Behar, S Hern and W Engelmann. The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. *Journal of Exposure Analysis and Environmental Epidemiology* 2001; 11: 231-52.

Lai, A, T Thatcher and W Nazaroff. Inhalation Transfer Factors for Air Pollution Health Risk Assessment. *Journal of the Air & Waste Management Association* 2000; 50: 1688-99.

Larsson, B, G Sahlberg, A Eriksson and L Busk. Polycyclic Aromatic Hydrocarbons in Grilled Food. *Journal of Agriculture and Food Chemistry* 1983; 31: 867-73.

Layton, D. Metabolically Consistent Breathing Rates for Use in Dose Assessments. *Health Physics* 1993; 64(1): 23-36.

Leopold Center. Food, Fuel, and Freeways: An Iowa Perspective on how Far Food Travels, Fuel Usage, and Greenhouse Gas Emissions. *Leopold Center for Sustainable Agriculture, June 2001. Report available for download at http://www.leopold.iastate.edu/* 2001.

Lioy, P, J Waldman, A Greenberg, R Harkov and C Pietarinen. The Total Human Environmental Exposure Study (THEES) to Benzo(a)pyrene: Comparison of the Inhalation and Food Pathways. *Archives of Environmental Health* 1988; 43(4): 304-312.

Mackay, D and E Webster. Linking Emissions to Prevailing Concentrations: Exposure on a Local Scale. *Environmetrics* 1998; 9: 541-553.

McDonald, J, B Zielinska, E Fujita, J Sagerbiel, J Chow and J Watson. Fine Particle and Gaseous Emission Rates from Residential Wood Combustion. *Environmental Science and Technology* 2000; 34(11): 2080-91.

McKone, T. CalTOX: A Multimedia Total Exposure Model for Hazardous Waste Sites. *UCRL-CR-111456PtI-IV. Lawrence Livermore National Laboratory Report, Livermore, CA*. Located at http://www.cwo.com/~herd1/caltox.htm. 1993.

McKone, T. Uncertainty and Variability in Human Exposures to Soil Contaminants through Home-grown Foods: A Monte Carlo Assessment. *Risk Analysis* 1994; 14: 449-63.

McKone, T, A Bodnar and E Hertwich. Development and Evaluation of State-Specific Landscape Data Sets for Life-Cycle Impact Assessment. *Research supported by: USEPA Sustainable Technology Division, National Risk Management Research Laboratory and the Environmental Defense Fund,* 1998.

McKone, T and J Daniels. Estimating Human Exposure through Multiple Pathways from Air, Water, and Soil. *Regulatory Toxicology and Pharmacology* 1991; 13: 36-61.

McKone, T, R Maddalena, DH Bennett and K Enoch. CalTOX : A Multimedia Total Exposure Model. *Available at: http://eetd.lbl.gov/ied/ERA/. Last accessed September 23, 2002.*

McLachlan, M. Bioaccumulation of Hydrophobic Chemicals in Agricultural Food Chains. *Environmental Science and Technology* 1996; 30(1): 252-59.

McLachlan, M. Framework for the Interpretation of Measurements of SOCs in Plants. *Environmental Science and Technology* 1999; 33(11): 1799-1804.

Microsoft. Streets98 (CD ROM) V 6.0. 1997.

Odabasi, M, N Vardar, A Sofuoglu, Y Tasdemir and T Holsen. Polycyclic aromatic hydrocarbons (PAHs) in Chicago air. *The Science of the Total Environment* 1999; 227: 57-67.

Santodonato, J, P Howard and D Basu. Health and Ecological Assessment of Polynuclear Aromatic Hydrocarbons. *Journal of Environmental Pathology and Toxicology* 1981; 5(1): 87, 122-127, 162-166.

Silveira, J. From the Pacific Coast FM Association. Personal Communication, March 1999.

Smith, D and R Harrison. Concentrations, Trends, and Vehicle Source Profile of Polynuclear Aromatic Hydrocarbons in the U.K. Atmosphere. *Atmospheric Environment* 1996; 30(14): 2513-25.

Smith, K, G Thomas and E Jones. Seasonal and Species Differences in the Air-Pasture Transfer of PAHs. *Environmental Science and Technology* 2001; 35(11): 2156-65.

Thomas, K, L Sheldon, E Pellizzari, R Handy, J Roberds and M Berry. Testing Duplicate Diet Sample Collection Methods for Measuring Personal Dietary Exposures to Chemical Contaminants. *Journal of Exposure Analysis and Environmental Epidemiology* 1997; 7(1): 17-36.

Tomerlin, J, M Berry, N Tran, S Chew, B Petersen, K Tucker and K Fleming. Development of a dietary exposure potential model for evaluating dietary exposure to chemical residues in food. *Journal of Exposure Analysis and Environmental Epidemiology* 1997; 7: 81-102.

USDA. Agricultural Handbook No. 8. *United States Department of Agriculture* 1979-1986.

USDA. 1989-91 Continuing Survey of Food Intakes by Individuals and 1989-91 Diet and Health Knowledge Survey on CD-ROM. *Available from NTIS. Accession number PB96-501747* 1996a.

USDA. Farmer's Market Survey Report. *Transportation and Marketing Division, Agricultural Marketing Service, 17 pps.* 1996b.

Vaessen, H, A Jekel and A Wilberg. Dietary Intake of Polycyclic Aromatic Hydrocarbons. *Toxicological and Environmental Chemistry* 1988; 16: 281-94.

Vaessen, H, P Schuller, A Jekel and A Wilberg. Polycyclic Aromatic Hydrocarbons in Selected Foods: Analysis and Occurrence. *Toxicological and Environmental Chemistry* 1984; 7: 297-324.

VanMetre, P, B Mahler and E Furlong. Urban Sprawl leaves its PAH signature. *Environmental Science and Technology* 2000; 34(19): 4064-70.

Voutsa, D and C Samara. Dietary Intake of trace elements and polycylic aromatic hydrocarbons via vegetables grown in an industrial Greek area. *The Science of the Total Environment* 1998; 218: 203-16.

Wagrowski, D and R Hites. Polycyclic Aromatic Hydrocarbon Accumulation in Urban, Suburban, and Rural Vegetation. *Environmental Science and Technology* 1997; 31(1): 279-82.

Wania, F and D Mackay. Tracking the Distribution of Persistent Organic Pollutants. *Environmental Science and Technology* 1996; 30(9): 390-96.

Welsch-Pausch, K and M McLachlan. Fate of airborne polychlorinated dibenzo-p-dioxins and dibenzofurans in an agricultural ecosystem. *Environmental Pollution* 1998; 102: 129-37.

Welsch-Pausch, K, M McLachlan and G Umlauf. Determination of the principal pathways of polychlorinated dibenzo-p-dioxins and dibenzofurans to Lolium Multiflorum (Welsh Ray Grass). *Environmental Science and Technology* 1995; 29(4): 1090-98.

Whicker, F and T Kirchner. Pathway: A Dynamic Food Chain Model to Predict Radionuclide Ingestion After Fallout Deposition. *Health Physics* 1987; 52: 717-37.

Wickstrom, K, H Pyysalo, S Plaami-Heikkilii and J Tuominen. Polycyclic aromatic compounds (PAC) in leaf lettuce. *Z Lebensim Upters Forsch* 1986; 183: 182-85.
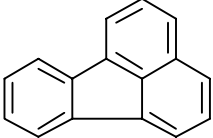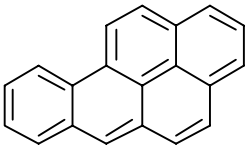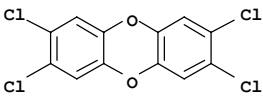
Wilson, K, J Chuang and C Lyu. Levels of persistent organic pollutants in several child day care centers. *Journal of Exposure Analysis and Environmental Epidemiology* 2001; 11: 449-58.

Zartarian, V, W Ott and N Duan. A quantitative definition of exposure and related concepts. *Journal of Exposure Analysis and Environmental Epidemiology* 1997; 7(4): 411-37.

Table 1: A selection of available food residue databases.

Database	Source
FDA Pesticide Program: Residue Monitoring 1993-99	(FDA, 2000)
California Pesticide Monitoring Database (CPMD)	(DPR, 2000)
Fish and Wildlife Residue Database 1995	(EPA, 2000)
MARCIS (Microbiological and Residue Computer Information Systems) Pesticide Residue Database 1990-95	(FSIS, 1995)
Pesticide Residue Information System, 1987-1994	(EPA, 1993)
Total Diet Study (TDS) Residue Database 1982-94	(FDA, 2001)
1994 Pesticide Data Program (PDP) Residue Database	(USDA, 2000)

Table 2: Physico-chemical and toxicological data for representative SVOC's, listed in order of increasing MW. The mean and coefficient of variation (CV) are given.

Chemical (formula) [CAS ID]	Structure	MW [g/mol]	VP[1] [Pa]	Log K_{ow}[1]	H [Pa-m^3/mol] [1]	Carc Class
Fluoranthene ($C_{16}H_{10}$) [206-44-0]		202	1.2E-03 (0.5)	5.1 (0.9)	1.0 (0.5)	D [2]
Benzo[a]pyrene ($C_{20}H_{12}$) [50-32-8]		252	7.1E-07 (0.1)	6.3 (1.0)	9.2E-02 (0.1)	B2 [2]
2,3,7,8-Tetrachloro dibenzo-p-dioxin ($C_{12}H_4Cl_4O_2$) [1746-01-6]		322	1.6E-06 (1.6)	6.7 (1.0)	2.5 (1.5)	A/B1 [3]

[1] (McKone, 1993)
[2] (EPA, 2001)
[3] (EPA, 2001)

Carc : = Carcinogenicity

Table 3: Summary of parameters used in (a) numerator and (b) denominator of the empirical $\theta_{ing/inh}$(fvg) for benzo(a)pyrene, reported as arithmetic mean and coefficient of variation (CV).

Table (3a) Summary of mean (CV) of parameters in the numerator of $\theta_{ing/inh}$(fvg).

j =	$\overline{C_{j,w}}$ [μg BaP/ kg j]	IR_j [kg j / kg-BW / d]
Exposed fruits	1.52 (0.4)	1.4E-03 (3.1)[1a]
Protected fruits	6.7E-02 (0.3)	1.7E-03 (1.7)[1b]
Exposed vegetable	8.3E-01 (0.9)	8.6E-04 (1.9)[1c]
Protected vegetable	1.14 (0.3)	3.3E-04 (23.0)[1d]
Root vegetable	6.5E-01 (0.7)	1.3E-03 (1.2)[1e]
Leafy vegetable	1.6 (0.9)	6.3E-04 (3.6E-03)[1f]
Grains	3.5E+01 (0.4)	4.1E-03 (8.8E+02)[1g]

Table (3b) Summary of mean (CV) of parameters in the denominator of $\theta_{ing/inh}$(fvg).

	Mean (CV)
C_{air} [ng/ m³]	
Outdoor [2-8]	6.0 (1.8)
Indoor [9]	1.1 (0.6)
BR [m3/ kg/ hr] [10]	1.5E-02 (0.3)

$\overline{C_{j,w}}$= intake weighted concentration of chemical (BaP) in food category i [μg/kg]. See appendix, Table A-1 for the summary and sources of individual concentrations of fvg's (C_i) within each food category, j.

IR_i := Intake rate (fresh weight basis) of food category i [kg i / kg-BW / d]

C_{air} := concentration of the chemical (BaP) in air [μg/m³]. If the average was not given, then estimated as midpoint from reported minimum and maximum concentrations reported. Since some PAH levels in outdoor air were given on a total, gaseous (PUF) and/or associated with particle phase PM, only used the total values. Where the measurements were not specified, assumed BaP concentration referred to total (gaseous+particle) basis.

BR := breathing rate [m3/kg(BW)/d]

[1] (EPA, 1997):
(a)Table 9-7, original source 1989-91 CSFII data (USDA, 1996a);
(b)Table 9-8, original source 1989-91 CSFII data (USDA, 1996a);
(c)Table 9-9, original source 1989-91 CSFII data (USDA, 1996a)and subtracting the leafy vegetables (from 8f) value still use same STDEV as from Table 9-9;
(d)Table 9-10, original source 1989-91 CSFII data (USDA, 1996a);
(e)Table 9-11, original source 1989-91 CSFII data (USDA, 1996a);
(f) original source USEPA, 1984b (Table 9_14 for total US population [g/d]); don't know n to convert SE to STDEV, so, use average STDEV from exposed, protected, and root vegetable. Also divide by lognormally distributed combined (adult and child) BW with arithmetic mean = 62.0 kg, CV= 0.2 (McKone, 1993) available W values were grouped according to food category i, and the average W, and CV, is reported here from (USDA, 1979-1986) as cited in (EPA, 1997), Table 9-27.
(g) Table 12-1, original source 1989-91 CSFII data on per capita intake of total grain (including mixtures).

[2] average concentration in winter urban air (Hawthorne et al., 1992).

[3] ARB, 1997 and ARB 1992 as cited in (CARB, 1997) monitored by the California Air Resources Board air toxics network.

[4] (Smith et al., 2001) average atmospheric concentration in semirural location.

[5] (Smith and Harrison, 1996) Urban site in summer and winter and rural site in summer and summary table of previous measurements.

[6] (Odabasi et al., 1999) Total (PM and gaseous) outdoor air concentrations of BaPin Chicago (measured) and summarized from previous research in Chicago, Houston, Boston and London.

[7] (Baek et al., 1991) Total (PM and gaseous) outdoor air concentrations of BaP in US urban regions and rural areas.

[8] (Coleman et al., 1997) Measured BaP concentration in summer and winter in London and Manchester, England.

[9] Sheldon et al (1993) and CARB (1992) as cited in (CARB, 1997); average indoor concentrations in smoking, wood burning and gas heat, and no source homes in Northern and Southern California.

[10] BR= 8/24* resting BR + 16/24 * active BR where mean(CV) of active BR is 1.90E-02 (0.3) m3 / kg/ hr and for resting BR, 6.40E-03 (0.2) m3/kg/hr from CalTOX (McKone, 1993); combined adult and child BR used. Assumed a body weight of 62 kg, as used in CalTOX (McKone, 1993).

Table A-1: Parameters used in calculating the intake weighted concentration, $C_{j,w}$ of benzo(a)pyrene [μg/ kg] in fruit, vegetable and grains from the literature. Whether the concentration, C_i, was reported on a dry weight (DW), fresh weight (FW), or undesignated weight (undes.) basis is also specified. The water content (W) and intake rate (IR_i) of each fvg (i) is also given. C_i references are given separately, as letters, below the footnotes.

j = Exposed Fruits

i =	Min C_i [μg/kg]	Max C_i [μg/kg]	$\overline{C_i}$'s [μg/kg]	C_i Reference	FW basis	DW basis	Undes. basis	W [%] [1]	IR_i [g/kg BW-d] [2]
Apple	2.0E-02	60	7.5E-01	a-e	1	4	3	84.2 [3]	4.6E-01
Grape	2.0E-02	0.2	2.8E-03	c, d			2	81.3	4.4E-02
Pear	5.0E-2	1.9	6.9E-02	c, d			2	83.8	1.2E-01
Persimmon			4.6E-06	d			1	85.0 [4]	4.0E-04
Plums	4.0E-02	29.7	2.1E-01	c, d			2	85.2	2.5E-02
Strawberry			ND	d			1	91.6	3.5E-02

j = Protected Fruits

i =	Min C_i [μg/kg]	Max C_i [μg/kg]	$\overline{C_i}$'s [μg/kg]	C_i Reference	FW basis	DW basis	Undes. basis	W [%] [1]	IR_i [g/kg BW-d] [2]
Banana	2.0E-02	0.16	1.9E-02	d, e	1		1	74.3	2.2E-01
Cantaloupe			4.4E-04	e	1			89.8	4.4E-02
Grapefruit			1.4E-03	e	1			90.9	6.9E-02 [6]
Oranges	3.0E-02	0.16	1.3E-02	d, e	1		1	86.8	1.5E-01 [6]
Orange Peel			1.2E-05	d			1	86.8 [5]	1.4E-04
Pineapple			3.6E-04	d			1	86.5	3.1E-02

j = Exposed Vegetables

i =	Min C_i [μg/kg]	Max C_i [μg/kg]	$\overline{C_i}$'s [μg/kg]	C_i Reference	FW basis	DW basis	Undes. basis	W [%] [1]	IR_i [g/kg BW-d] [2]
Cucumber			ND	f			1	96.1	7.2E-02
Eggplant			ND	f			1	91.9	6.2E-03
Mushroom			8.6E-02	c			1	91.8	2.1E-02
Onion Greens			1.1E-04	g			1	93.0 [7]	2.0E-03
Tomatoes	1.0E-02	6.65	4.1E-01	a, h, i, e, j	1	2	2	94.0	4.9E-01

j = Protected Vegetables

i =	Min C_i [μg/kg]	Max C_i [μg/kg]	$\overline{C_i}$'s [μg/kg]	C_i Reference	FW basis	DW basis	Undes. basis	W [%] [1]	IR_i [g/kg BW-d] [2]
Corn (above ground)			2.9E-01	k			1	76.0	2.4E-01
Kidney Bean			ND	f			1	80.3 [8]	1.4E-02
Pumpkin			ND	g			1	91.6	4.4E-03
Soya Beans			ND	a			2	69.1	

j = Root Vegetables

i =	Min C_i [μg/kg]	Max C_i [μg/kg]	$\overline{C_i}$'s [μg/kg]	C_i Reference	FW basis	DW basis	Undes. Basis	W [%] [1]	IR_l [g/kg BW-d] [2]

i =	Min C_i [µg/kg]	Max C_i [µg/kg]	$\overline{C_i}$'s [µg/kg]	C_i Reference	FW basis	DW basis	Undes. basis	W [%] [1]	IR_i [g/kg BW-d] [2]
Beet	2.0E-01	0.21	3.1E-03	l		2		87.3	2.2E-01
Carrot	ND	0.22	8.2E-03	f, e, l, y	2	1	1	87.8	1.7E-01
Endive	1.29E-02	50	3.7E-03	m, l, n, y	2	2		93.8	1.1E-03
Leek	7.5E-03	6.6	3.9E-05	j, l, y	1	1	2	83.0	3.9E-05
Onion Bulb	ND	7.36	1.5E-02	g, l	2	2	1	90.8	1.1E-01
Potatoes	ND	23.49	1.0E+00	h, g, o, e	1	9	1	81.1 [9]	1.1E+00 [10]
Radish roots	ND	1.2	4.8E-05	h, g		1	1	94.8	1.6E-03
Sweet potatoes	ND	0.17	3.3E-03	g, e	1		1	72.8	3.9E-02

j = Leafy Vegetables

i =	Min C_i [µg/kg]	Max C_i [µg/kg]	$\overline{C_i}$'s [µg/kg]	C_i Reference	FW basis	DW basis	Undes. basis	W [%] [1]	IR_i [g/kg BW-d] [2]
Broccoli			8.3E-03	e	1			90.7	4.9E-02
Cabbage	ND	24.5	9.7E-02	h, g, o, p, l, y	1	13	2	91.3 [11]	9.4E-02
Chinese Cabbage			1.3E-04	g			1	95.3	4.6E-03
Cauliflower	1.2E-01	5.1	2.4E-02	c, e	1		1	92.3	1.6E-02
Collard Greens			9.1E-03	e	1			93.9	1.9E-02
Kale	4.7E-01	48.6	1.3E-02	a, e, q, r,-t	4		3	84.5	1.5E-03
Lettuce	ND	150	4.9E-01	a, h, m, u-w, y	10	15	4	95.4 [12]	2.3E-01 [13]
Mustard Greens			1.9E-03	e	1			90.8	1.5E-02
Parsley leaf and Stem (tops)			5.2E-02	h			1	88.3	3.7E-03
Spinach	9.0E-02	20	5.5E-02	e, f, h, j, m, s	2	2	2	91.6	4.4E-02
Turnip Greens			1.5E-03	e	1			91.1	1.5E-02

j = Grains

i =	Min C_i [µg/kg]	Max C_i [µg/kg]	$\overline{C_i}$'s [µg/kg]	C_i Reference	FW basis	DW basis	Undes. basis	W [%] [1]	IR_i [g/kg BW-d] [2]
Barley- grain	3.0E-01	4.5	3.0E-01	e, t	3	3		10.1	1.5E-01 [15]
Oats-grain	2.0E-01	4.6	1.8E-01	k		2		8.2	8.3E-02
Rye			8.7E-03	x	1			10.4	4.3E-03 [16]
Wheat- grain	1.0E-01	540	5.6E+01	j, k, x	1	2	2	10.2 [14]	1.4E+00 [17]
SUM of FW / DW / undes.					41	63	48		

(1) average water content in edible portion of food ((EPA, 1997); Table 9-27 for f&v's and Table 12-21 for grain) in order to convert from DW to FW concentration.

(2) 'as consumed mean per capita intake rates' ((EPA, 1997); from Table 9-13 for f&v's and Table 12-12 for grains, original source 1989-91 CSFII)

(3) assume average W of apples with and without skin ((EPA, 1997); Table 9-27)

(4) since W not reported for persimmon's, assume the average of W's for exposed fruits ((EPA, 1997); Table 9-27)

(5) assume same water content as the 'oranges'- unspecified ((EPA, 1997); Table 9-27)

(6) pulp only intake ((EPA, 1997); Table 9-13, original data from DRES 1977,78)

(7) since W not specified for onion greens, assume the average of W's for exposed vegetables as reported in ((EPA, 1997); Table 9-27)

(8) average W of lima and snap beans ((EPA, 1997); Table 9-27)

(9) average W of potatoes (White) peeled and potatoes (white) whole from ((EPA, 1997); Table 9-27)

(10) sum of all potato related intake ((EPA, 1997); Table 9-13, data originally from DRES, 1977,78)

(11) average W of red and savoy cabbage ((EPA, 1997); Table 9-27)

(12) average W of iceberg and romaine lettuce ((EPA, 1997), Table 9-27)

(13) intake of all lettuce varieties ((EPA, 1997); Table 9-13 original data from DRES 1977,78)

(14) average of all W's for wheat related items ((EPA, 1997); Table 12-21)

(15) assume average intake of all grain intake ((EPA, 1997); Table 12-12, original data from DRES 1977-78 NFCS)

(16) sum of rye- rough, rye-germ, and rye-flour ((EPA, 1997); Table 12-12, original data from DRES 1977-78 NFCS)

(17) sum of rough, germ, bran and flour wheat intake ((EPA, 1997); Table 12-12, original data from DRES NFCS 1977,78)

[a]   Hettche (1971), Fritz and Engst (1971), Grimmer and Duvel (1970) and Grimmer and Hildebrandt (1965), as cited in (IARC, 1973)

[b]   Fritz (1971) in (Edwards, 1983)

[c]   Kolar et al (1975) in (Santodonato et al., 1981)

[d]   Shiraishi et al (1975) in (Santodonato et al., 1981)

[e]   (Kazerouni et al., 2001)

[f]   Shiraishi et al (1973), as cited in (Santodonato et al., 1981)

[g]   Shiraishi et al (1974) in (Santodonato et al., 1981)

[h]   Kolar et al (1975) as cited in (Santodonato et al., 1981) and (Edwards, 1983)

[i]   Wang and Meresz (1981) in (Edwards, 1983)

[j]   (Grimmer and Hildebrandt, 1965b)

[k]   Kolar et al (1975) in (Edwards, 1983)

[l]   (Voutsa and Samara, 1998)

[m]   Graf and Diehl (1966) as cited in (Edwards, 1983)

[n]   Prinsen 1979 in (Vaessen et al., 1984)

[o]   Shkodich and Litvinov (1979) in (Edwards, 1983)

[p]   Sokolowska (1980) in (Edwards, 1983)

[q]   Grimmer (1981), as cited in (Vaessen et al., 1984)

[r]   (Vaessen et al., 1984)

[s]   (Vaessen et al., 1988)

[t]   Hetteche (1971) in (Santodonato et al., 1981)

[u]   (Larsson et al., 1983)

[v]   (Wickstrom et al., 1986)

[w]   Larsson and Sahlberg (1981) in (Edwards, 1983)

[x]   (Grimmer and Hildebrandt, 1965a)

[y]   (Kipopoulou et al., 1999)
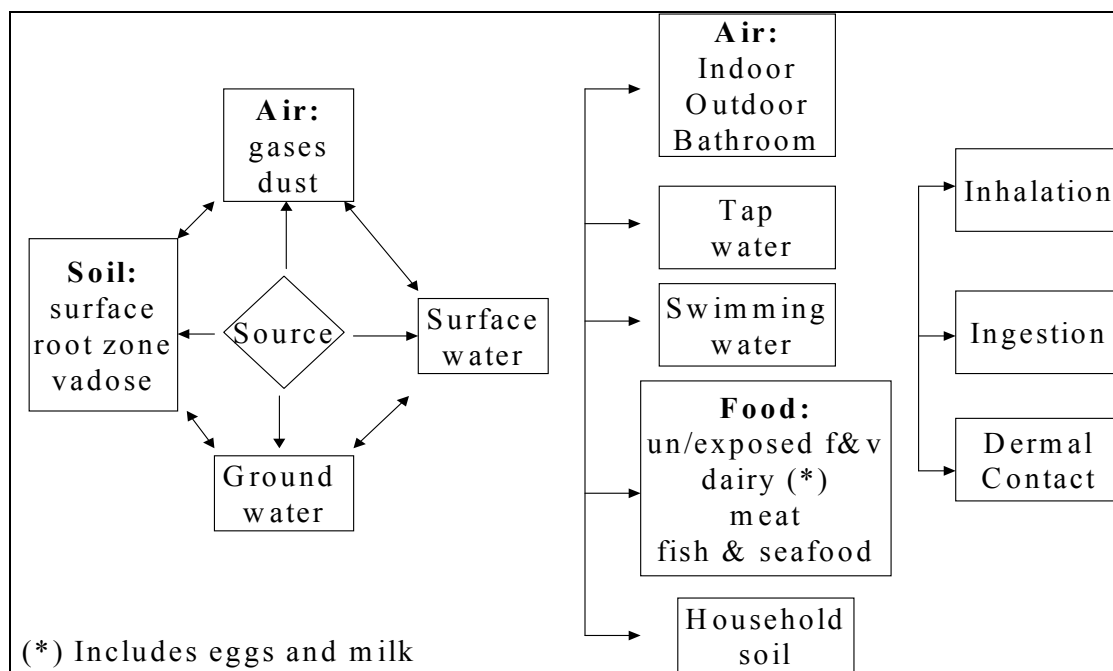
Figure 1: Pathways included in the CalTOX multimedia model framework (adapted from (McKone and Daniels, 1991). Source value was 1 g SVOC / d emitted to air.
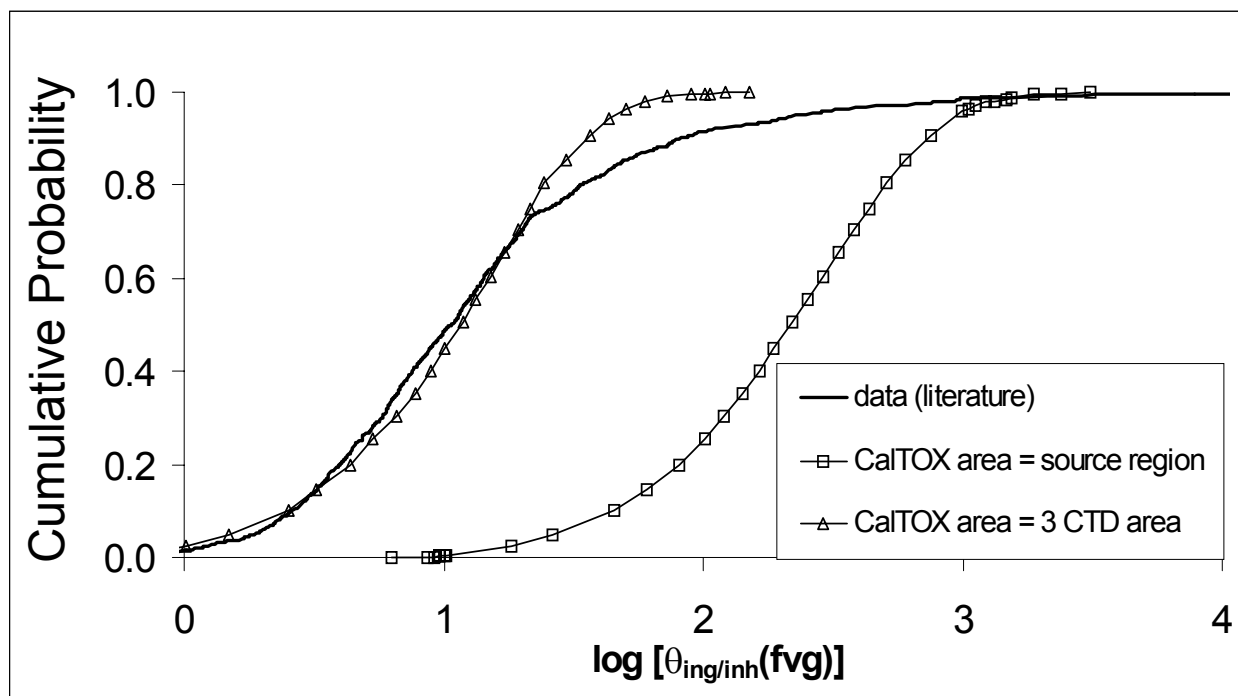
Figure 2: Comparison of the empirical $\theta_{ing/inh}$(fvg) for benzo(a)pyrene derived from data reported in the archived literature and the CalTOX multimedia model. Cumulative probability for the CalTOX model simulations is based on 1,000 Monte Carlo simulations assuming lognormal distributions for relevant human exposure and fate and transport parameters.
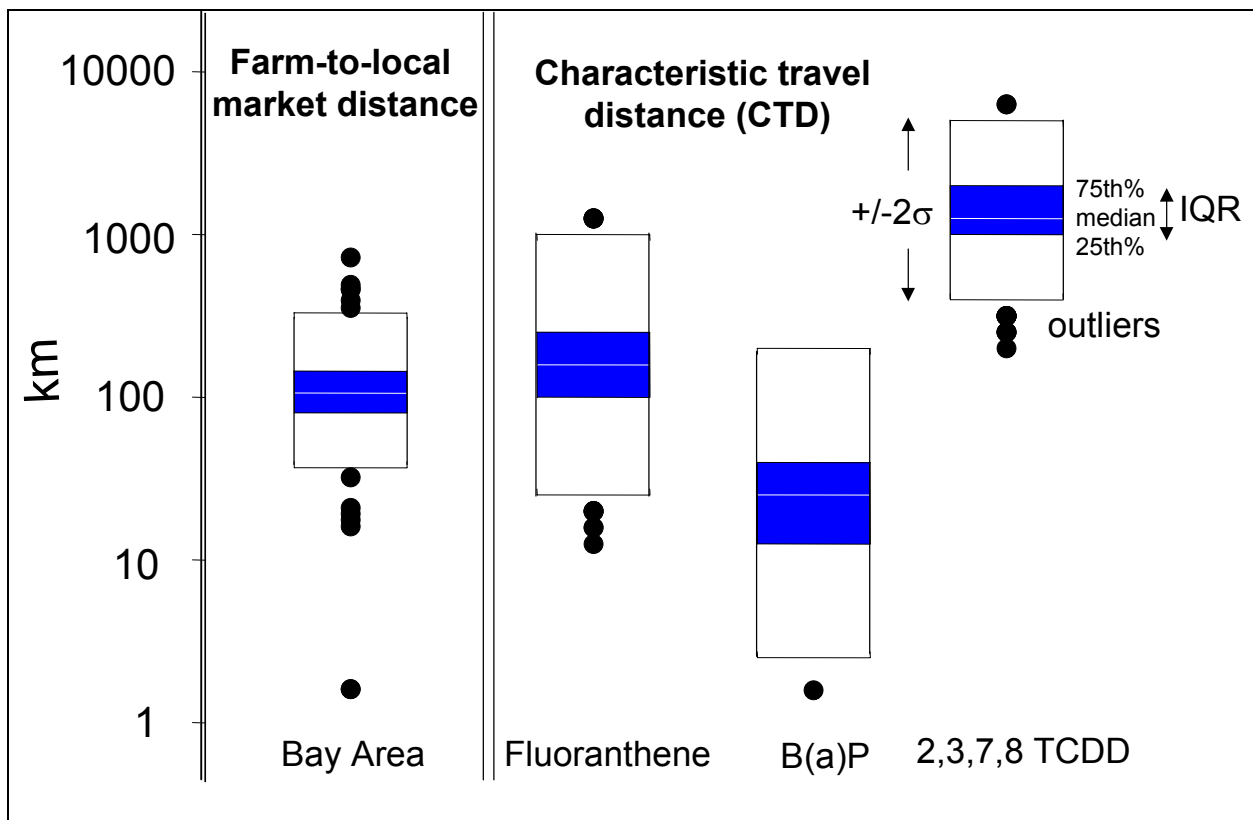
Figure 3: Farm-to-local market distance (for the San Francisco Bay Area) and characteristic travel distance (CTD) , for selected SVOC's. IQR is the interquartile range. CTD's were simulated using the CalTOX model.
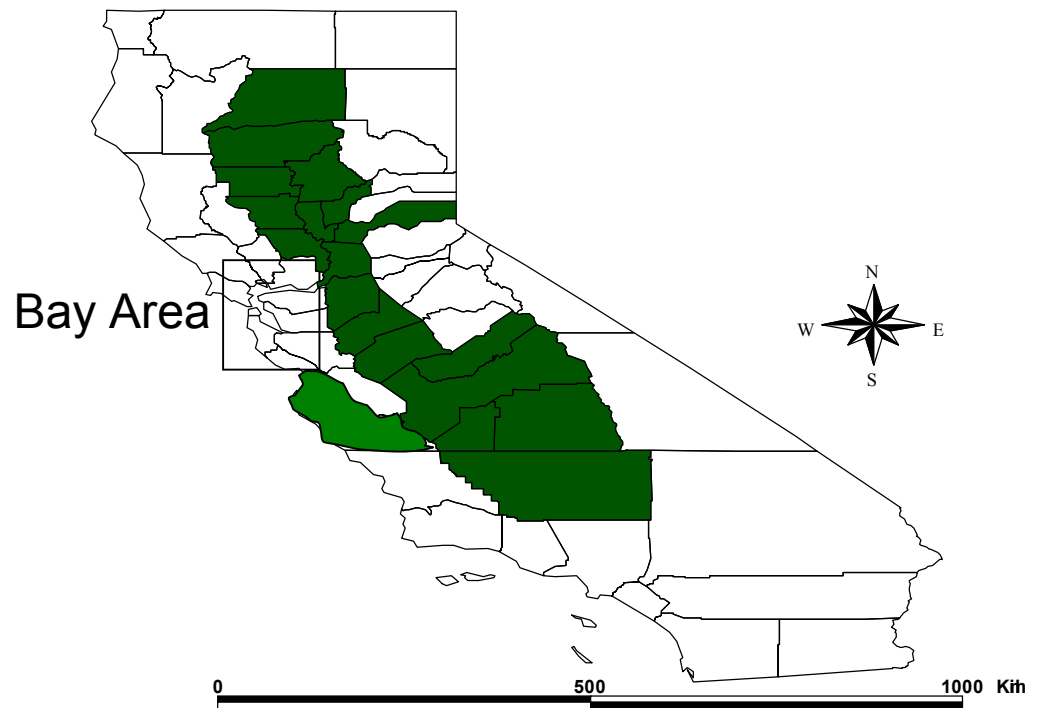
Figure 4: The San Francisco Bay Area in relation to the Central and Salinas Valleys of California.
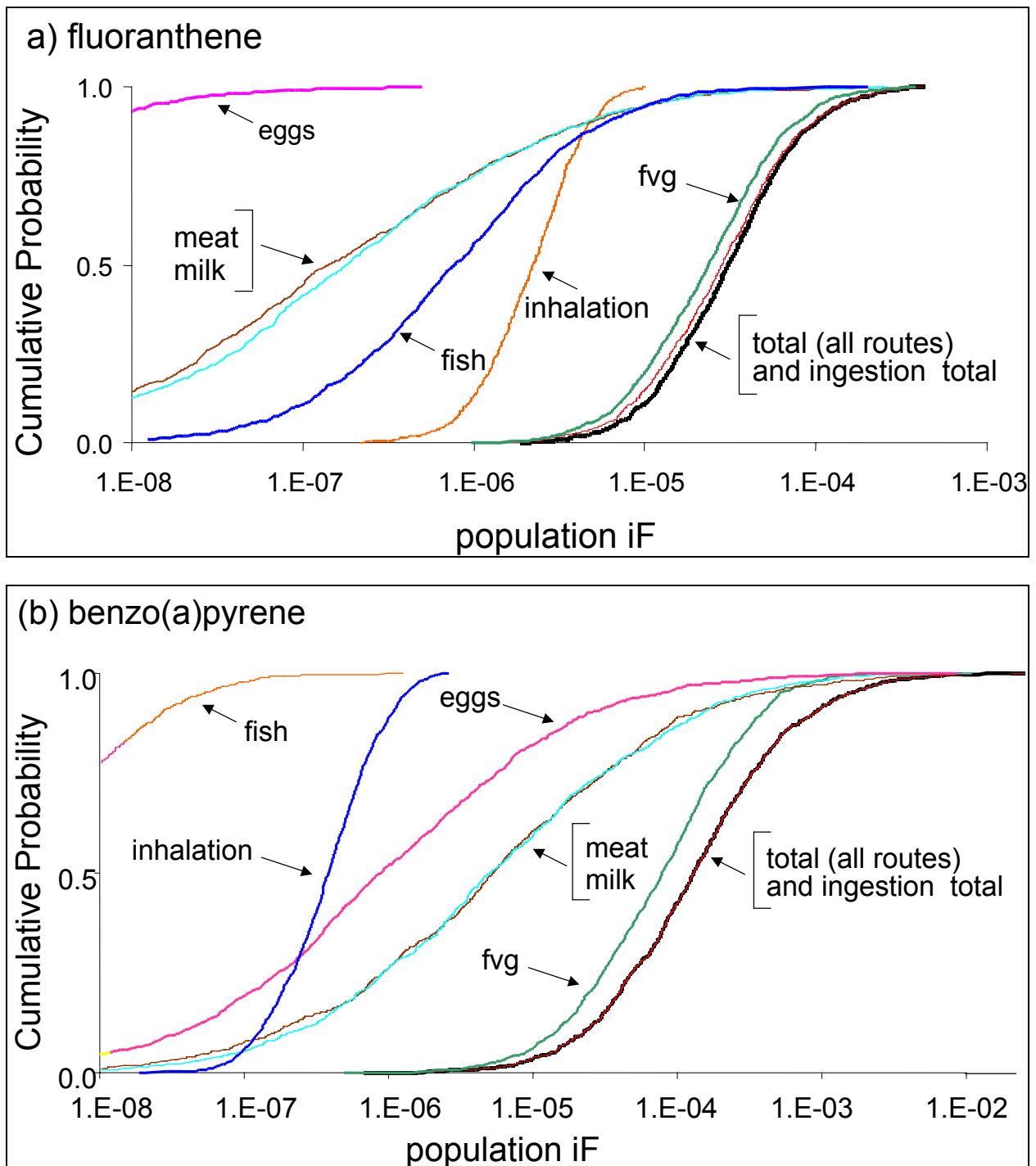
Figure 5: Cumulative density functions of pathway specific log population based iFs using California meteorological & landscape parameters and the area of the San Francisco Bay Area. Local food contribution was set at 100% for a) fluoranthene and b) benzo(a)pyrene. For both figures, fvg dominates as a pathway specific contributor to intake.